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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/696,909	10/29/2003	James B. Lorens	7946-79836-01	9257
74839	7590	06/23/2008	EXAMINER	
Klarquist Sparkman, LLP 121 SW Salmon St Floor 16 Portland, OR 97204				REDDIG, PETER J
ART UNIT		PAPER NUMBER		
1642				
			MAIL DATE	DELIVERY MODE
			06/23/2008	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/696,909	LORENS ET AL.	
	Examiner	Art Unit	
	PETER J. REDDIG	1642	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1) Responsive to communication(s) filed on 12 March 2008.

2a) This action is **FINAL**. 2b) This action is non-final.

3) Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4) Claim(s) 1,6,12,14-18,27,41-44 and 54 is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5) Claim(s) _____ is/are allowed.

6) Claim(s) 1, 12, 14-18, 27, 41-44 and 54-6 is/are rejected.

7) Claim(s) _____ is/are objected to.

8) Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9) The specification is objected to by the Examiner.

10) The drawing(s) filed on _____ is/are: a) accepted or b) objected to by the Examiner.

 Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

 Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11) The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12) Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a) All b) Some * c) None of:

- Certified copies of the priority documents have been received.
- Certified copies of the priority documents have been received in Application No. _____.
- Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) Notice of References Cited (PTO-892)

2) Notice of Draftsperson's Patent Drawing Review (PTO-948)

3) Information Disclosure Statement(s) (PTO/SB/08)
Paper No(s)/Mail Date _____.

4) Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____.

5) Notice of Informal Patent Application

6) Other: _____.

DETAILED ACTION

1. The Amendment filed March 12, 2008 in response to the Office Action of December 12, 2007 is acknowledged and has been entered. Previously pending claims 1, 27 and 55 have been amended, and new claims 56-61 have been added. Claims 1, 12, 14-18, 27, 41-44 and 54-61 are currently being examined.
2. The Declaration of Dr. Sacha Holland under 37 CFR 1.132 filed March 12, 2008 is sufficient to overcome the rejection of claims 1, 12, 14-18, 27, 41-44 and 55 based upon their rejection under 35 U.S.C. 112, first paragraph for the reasons set forth in the Office Action of December 12, 2007, section 7-10, page 4-26.
3. The following rejections are being maintained:

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

4. Claims 1, 12, 14-18, 27, 41-44 and 55 remain rejected and new claims 56-61 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement, for the reasons set forth in the Office Action of December 12, 2007, section 11, pages 26-29.

In the Office Action of December 12, 2007, Examiner argued:

Currently amended claim 1, and thus its dependent claims, have no clear support in the specification and the claims as originally filed. Applicants argue that support for amended Claim 1 is found throughout the specification, for example, at page 6, lines 7-20. Claim 1 is also

amended to recite performance of assays in the presence and absence of the test compound.

Support for this amendment is found throughout the specification, for example, at page 9, line 32 through page 10, line 4. Claim 1 is also amended to recite a step of performing a cell based angiogenesis phenotype assay using a cell that comprises the Axl angiogenesis polypeptide.

Support for this amendment is found throughout the specification, for example, at page 8, lines 18-24, page 30, lines 6-10 and 14-29, page 31, line 22 through page 33, line 12, and page 32, lines 25-26. Claim 1 is further amended to recite that inhibition of Axl kinase activity and inhibition of the cell- based angiogenesis phenotype assay in the presence of the compound identify the compound as an inhibitor of angiogenesis. Support for this amendment is found throughout the specification, for example, at Figures 12-17, which demonstrate that an RNAi molecule specific for the nucleic acid that encodes the Axl angiogenesis polypeptide down regulates expression of the Axl polypeptide in a cell and that down regulation and lack of expression of the Axl polypeptide causes inhibition of cell-based angiogenesis assays. As expression of the Axl polypeptide is down regulated, kinase activity of the Axl polypeptide is also necessarily down regulated in the cell.

A review of the specification discloses support for Axl, its ligands, expression, and association with diseases (page 6, lines 7-20), a general description of an assay to identify inhibitors of angiogenesis/tumor genesis (page 9, line 32 through page 10, line 4), the definition of “functional effect” described *supra* (page 8, lines 18-24), a general description of assessing the modulation of an angiogenesis protein (page 30, lines 6-10), numerous assays to measure angiogenesis associated with tumors, tumor growth, neovascularization, endothelial tube 25 formation, cell surface markers such as alpha V beta 3, hormone release, transcriptional changes

to both known and uncharacterized genetic markers (e.g., northern blots), changes in cell metabolism such as cell growth or pH changes, and changes in intracellular second messengers such as cGMP. In one embodiment, measurement of integrin cell surface expression and FACS sorting is used to identify modulators of angiogenesis, (page 30, lines 14-29), measuring ligand binding, cell surface marker expression, cellular proliferation, VEGF-R assays, co-culture assays for tube formation, cell migration assays, mRNA or protein expression, haptotaxis, tube formation, CAM assays, cellular morphology (e.g., cell volume, nuclear volume, cell perimeter, and nuclear perimeter), ligand binding, kinase activity, apoptosis, cell surface marker expression, cellular proliferation, GFP positivity and dye dilution assays (e.g., cell tracker assays with dyes that bind to cell membranes), DNA synthesis assays, and cell cycle arrest, (page 31, line 22 through page 33, line 12, and page 32, lines 25-26), treatment of HUVEC cells with RNAi directed to Axl inhibits the haptotaxis, proliferation, and tube formation in HUVEC cells *in vitro* (figures 12-17) The suggested support is not found persuasive because there is nothing in the specification to suggest the specific combination of assay steps in claim 1 to identify a compound that inhibits angiogenesis.

Additionally, the teachings of the specification do not support an Axl polypeptide with “greater than” 95% identity to full length SEQ ID NO: 4, only “greater than about” 95% identity as described in claims 1 and 27, para. bridging p. 7 and 8. Furthermore, there is nothing in the specification or claims as originally filed to support an Axl polypeptide comprising SEQ ID NO:4, which encompasses sequences outside of SEQ ID NO: 4.

Thus the subject matter claimed in claims 1, 12, 14-18, 27, 40-44, 54 and 55 broadens the scope of the invention as originally disclosed in the specification and claims as originally filed.

Applicants argue that as established in *Ex parte Parks*, "adequate description under the first paragraph of 35 U.S.C. 112 does not require literal support for the claimed invention Rather, it is sufficient if the originally-filed disclosure would have conveyed to one having ordinary skill in the art that an appellant had possession of the concept of what is claimed" *Ex parte Parks*, 30 USPQ2d 1234, 1236-37 (B.P.A.I. 1993) (emphasis added). Moreover, the M.P.E.P. at §2163 states that "[w]hat is conventional or well known to one of skill in the art need not be disclosed in detail. See *Hybritech Inc. v. MonoclonalAntibodies, Inc.*, 802 F.2d at 1384, 231 USPQ at 94... If a skilled artisan would have understood the inventor to be in possession of the claimed invention at the time of filing, even if every nuance of the claims is not explicitly described in the specification, then the adequate description requirement is met. See, e.g. *Vas-Cath*, 935 F.2d at 1563, 19 USPQ2d at 1116; *Martin v. Johnson*, 454 F.2d 746, 751,172 USPQ 391,395 (CCPA 1972) (stating "description need not be in *ipsis verbis* [i.e., "in the same words"] to be sufficient")."

Applicants argue that the specification describes methods for identifying a compound that inhibits angiogenesis comprising contacting the compound with an Axl polypeptide and determining the functional effect of the compound on the polypeptide (page 2, lines 23-33). Determining the functional effect includes "assaying for a compound that increases or decreases a parameter that is indirectly or directly under the influence of an angiogenesis peptide" and includes enzymatic activity and cell-based angiogenesis phenotypes (including cell proliferation,

cell surface marker expression, haptotaxis, and tube formation) (page 8, line 27 to page 9, line 8). Further, assays for identifying modulators of angiogenesis proteins are described on page 30, lines 5-29. The specification states that "any suitable physical, chemical, or phenotypic change that affects activity or binding can be used to assess the influence of a test compound on the polypeptide of this invention" (page 30, lines 20-22). There is no suggestion that only one of the described assays may be used to identify an inhibitor of angiogenesis. Based on the specification, one of skill in the art would understand that a combination of the described assays may be desirable in identifying a compound that inhibits angiogenesis. In fact, the specification shows examples of the use of a combination of assays in Figure 13 (effect of Axl RNAi on haptotaxis and integrin expression) and Figure 15 (haptotaxis and cell proliferation).

Applicants argue that the specification describes both in vitro (both test-tube and cell-based) kinase assays and cell-based angiogenesis phenotype assays. Further, the specification describes the use of combinations of other assays. One of skill in the art would readily recognize from the specification that any combination of the described assays could be used in methods for identifying compounds that inhibit angiogenesis.

Applicants argue that in addition, the rejection asserts that there is no support in the claims or specification as originally filed to support "an Axl polypeptide comprising SEQ ID NO: 4, which encompasses sequences outside of SEQ ID NO: 4" (Office action, page 28, first full paragraph). Applicants argue that the specification describes labels which may be incorporated into a protein (page 16, lines 6-11) and fusion proteins (page 16, lines 24-26 and page 27, lines 20-25). Applicants argue that therefore, there is support in the specification for an Axl polypeptide which comprises SEQ ID NO: 4 and additional elements.

Applicant arguments have considered, but have not been found persuasive because the suggested support is not found persuasive because there is nothing in the specification to suggest the specific assay steps in claims 1, 27, and 56 to identify a compound that inhibits angiogenesis. The general description of determining the functional effects of the compound on Axl on (page 2, lines 23-33) and (page 8, line 27 to page 9, line 8) do not provide support for the specifically claimed method steps because there is no description of the combined method steps of claims 1 or any description of assaying the Axl kinase activity for identifying compounds that inhibit angiogenesis. Furthermore the general individual assays for identifying modulators of angiogenesis proteins are described on page 30, lines 5-29 do not provide support for the specifically claimed method steps because there is no description of the combined method steps of claims 1 or any description of assaying the Axl kinase activity for identifying compounds that inhibit angiogenesis. Figure 13 (effect of Axl RNAi on haptotaxis and integrin expression) and Figure 15 (haptotaxis and cell proliferation) do not provide support for the base claims because they do not teach either the combination methods of claims 1 or the assaying Axl kinase activity. Additionally, the general description of fusion proteins and labels does not provide support for the specific genus of Axl polypeptides comprising an amino acid sequence with greater than 95% identity to full length SEQ ID NO: 4 claimed in claims 1, 27, and 56.

Additionally, on page 5 of the remarks of the Remarks of March 12, 2008, Applicants point to support at:

Claim 1: page 5, lines 1-17; Figures 2, 12, 13, 15, and 17

Claim 27: page 5, lines 1-17; page 16, lines 12-18; page 30, line 5 to page 31, line 32; Figures 2, 12, 13, 15, and 17

Claim 56: page 16, lines 12-18; page 31, lines 23-32

Claims 57-60: page 2, lines 17-21; page 34, lines 24-29

Claim 61: page 5, lines 22-23

A review of the specification discloses support for a screening protocol for haptotactic migration inhibitors, a cellomics haptotaxis assay, that a CD13/N-aminopeptidase is involved in haptotaxis, that a transgluaminase II protein is involved in haptotaxis, that a zip kinase protein is involved in haptotaxis, that a PRK-1 protein is involved in haptotaxis, that PRK1 mRNA is expressed in endothelial cells and PBMCs, that a PRK-1 protein is involved in haptotaxis and alpha V beta 3 expression , PRK-1 RNAi reduces PRK-1 message, haptotaxis and alpha V beta 3 expression (page 5, lines 1-17 and Figure 2); AxL RNAi reduces Ax1 mRNA expression (Fig. 12); that Ax1 RNAi inhibit haptotaxis to vitronectin (figure 13); Ax1 RNAi inhibits vitronectin haptotaxis and HUVEC proliferation (figure 15); Ax1 RNAi inhibits tube formation in a co-culture assay (Fig. 17); a definition of the term recombinant (page 16, lines 12-18); a general description of assays for modulators of angiogenesis or tumorigenesis proteins (page 30, line 5 to page 31, line 32); methods of screening for compounds, e.g., small organic molecules, antibodies, nucleic acids, peptides, cyclic peptides, nucleic acids, antisense molecules, RNAi, and ribozymes, that are capable of modulating angiogenesis or tumorigenesis, e.g., either activating or inhibiting angiogenesis or tumorigenesis (page 2, lines 17-21); the compounds tested as modulators of the angiogenesis protein can be any small organic molecule, or a biological entity, such as a protein, e.g., an antibody or peptide, a sugar, a nucleic acid, e.g., an antisense oligonucleotide, RNAi molecule, or a ribozyme, or a lipid. Alternatively, modulators can be genetically altered versions of an angiogenesis protein. Typically, test compounds will be

small organic molecules, peptides, lipids, and lipid analogs; page 34, lines 24-29), the identification of Axl as angiogenesis/tumorigenesis polypeptide (page 5, lines 22-23).

The suggested support is not found persuasive because there is nothing in the specification to suggest the specific method steps of claims 1 or any description of assaying the Axl kinase activity for identifying compounds that inhibit angiogenesis or the genus of Axl polypeptides comprising an amino acid sequence with greater than 95% identity to full length SEQ ID NO: 4.

Applicant's arguments have not been found persuasive and the rejection is maintained.

New Grounds of Rejection

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

5. Claims 1, 12, and 14-18 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 1, 12, and 14-18 are rejected under 35 U.S.C. 112, second paragraph, as being incomplete for omitting essential steps, such omission amounting to a gap between the steps. See MPEP § 2172.01. The omitted steps are: contacting the Axl polypeptide or cell comprising the Axl polypeptide with the compound being examined.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

6. Claims 1, 14, 27, 54-56 and 61 are rejected under 35 U.S.C. 102(b) as being anticipated by Healy et al. (Am. J. of Physiology, Lung Cell Molecular Physiology, June, 2001 280: L1273-L1281, previously cited).

Healy et al. teaches determining the *in vitro* kinase activity of an Axl polypeptide where the Axl polypeptide has kinase activity in the absence of the compound, see Fig. 5 and page L1276, 2nd col. Healy et al. teaches performing a cell-based assay in an endothelial cell by contacting human pulmonary endothelial cells that express human Axl (see Fig. 2) with the Axl ligand Gas 6 and determining the effect of this interaction on cell number, see Abstract, p. 1276, left column, and Fig. 6. Healy et al. teaches assaying apoptosis in human endothelial cells expressing recombinant wild type Axl, see p. L1278 and Figure 9 and 10.

It is noted that the specification teaches assaying increases or decreases in cellular proliferation and apoptosis as cell based assays for assaying the effect of the test compounds and are among the “functional effects” contemplated for assaying the potential inhibitor, see page 8, line 15-to page 9, line 5. Thus “determining the functional effects of the compound upon the kinase activity of the Axl polypeptide”, when given its broadest reasonable interpretation, encompasses assaying cellular responses such as increases or decreases in cellular proliferation and apoptosis

It is noted that a wherein clause in a method claim is not given weight when it simply expresses the intended result of a process step positively recited, MPEP 2111.04. Given that the method of the prior art comprises the same method steps as claimed in the instant invention, determining, *in vitro* kinase activity of an Axl polypeptide comprising an amino acid sequence

with greater than 95% identity to full length SEQ ID NO: 4, wherein the Axl polypeptide has kinase activity in the absence of said compound; and performing a cell based assay in an endothelial cell comprising said Axl polypeptide, which assay produces an angiogenesis phenotype in said endothelial cell in the absence of the compound, the claimed method is anticipated because the method will inherently be a method for identifying a compound that inhibits angiogenesis, wherein inhibition of the in vitro kinase activity of the Axl polypeptide in the presence of the compound and inhibition of the angiogenesis phenotype in the cell-based assay in the presence of the compound identifies the compound as a compound that inhibits angiogenesis, wherein inhibition of the angiogenesis phenotype in the cell-based assay in the presence of the compound identifies the compound as a compound that inhibits angiogenesis, wherein inhibition of the angiogenesis phenotype in the cell-based assay is caused by down regulation of expression of the Axl polypeptide, or wherein inhibition of the kinase activity of the Axl polypeptide in the presence of the compound identifies the compound as a compound that inhibits angiogenesis. See Ex parte Novitski 26 USPQ 1389 (BPAI 1993). Although the reference does not specifically state that the method is a method for identifying a compound that inhibits angiogenesis, the claimed method appears to be the same as the prior art method, absent a showing of unobvious differences. The office does not have the facilities and resources to provide the factual evidence needed in order to establish that the method of the prior art does not possess the same material, structural and functional characteristics of the claimed method. In the absence of evidence to the contrary, the burden is on the applicant to prove that the claimed method is different from those taught by the prior art and to establish patentable differences. See *In re Best* 562F.2d 1252, 195 USPQ 430 (CCPA).

Although the reference does not specifically state that the Axl of the reference is SEQ ID NO: 4 given that the Axl polypeptide of SEQ ID NO: 4 and Healy et al. are human Axl, the claimed product appears to be the same as the prior art product, absent a showing of unobvious differences. The office does not have the facilities and resources to provide the factual evidence needed in order to establish that the product of the prior art does not possess the same material, structural and functional characteristics of the claimed product. In the absence of evidence to the contrary, the burden is on the applicant to prove that the claimed product is different from that taught by the prior art and to establish patentable differences. See *In re Best*, 562 F2nd 1252, 195 USPQ 430 (CCPA 1977).

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

The factual inquiries set forth in *Graham v. John Deere Co.*, 383 U.S. 1, 148 USPQ 459 (1966), that are applied for establishing a background for determining obviousness under 35 U.S.C. 103(a) are summarized as follows:

1. Determining the scope and contents of the prior art.
2. Ascertaining the differences between the prior art and the claims at issue.
3. Resolving the level of ordinary skill in the pertinent art.
4. Considering objective evidence present in the application indicating obviousness or nonobviousness.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various

claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

7. Claims 12, 15-18, 41-44, and 57-60 are rejected under 35 U.S.C. 103(a) as being unpatentable over Healy et al. (Am. J. of Physiology, Lung Cell Molecular Physiology, June, 2001 280: L1273-L1281, previously cited) as applied to claims 1, 14, 27, 54-56 and 61, above, in view of Varner and Cheresh (Current Opinion in Cell Biology, October 1996, 8:724-730, previously cited), in further view of Ruoslahti et al (US Patent 6,180,084 January, 2001, previously cited), in further view of Panzer et al. (United States Patent Application Publication No.: 2004/0048253, February 21, 2001, previously cited), and in further view of Klinghoffer et al. (United States Patent Application Publication No.: 2004/0077574, May 23, 2002) .

Healy et al. teach as described *supra*.

Healy et al. do not teach determining the functional effect by measuring $\alpha V\beta 3$ expression or haptotaxis or the use of an antibody, an antisense molecule, an RNAi molecule, or a small organic molecule.

Varner and Cheresh teach that integrin $\alpha V\beta 3$ is significantly upregulated on vascular cells within human tumors and in response to growth factors and plays a biological role in a critical event of blood vessel formation during tumor angiogenesis, see section on Role of Integrins in Tumor Angiogenesis, p. 726- 727.

Panzer et al. teach the common art practice of screening small molecules, antibodies, oligonucleotides, and the antisense molecules for use in diagnosis and therapies, see para 0735-0741 and 0754-0757 of the published application. Similarly Ruoslahti et al teach the common art practice of screening organic chemicals, nucleic acid molecules such as RNA, a cDNA, or oligonucleotides, and antibodies for use in therapies, see Col 1, 2, 9-12.

Klinghoffer et al. teach that siRNA polynucleotides offer advantages over other types of polynucleotides for sequence specific alteration of gene expression including lower effective siRNA polynucleotide concentration, enhance stability, shorter lengths, they are readily taken up by intact cells, and are effective at concentration that are several orders of magnitude lower than those required for either antisense or ribozyme polynucleotides, see paragraph 0025.

It would have been *prima facie* obvious at the time the invention was made to perform the method of claim 1 by measuring α V β 3 expression and to use an antibody, antisense molecule, RNAi, or small organic molecule as the compound to use in the screening methods for claims 1, 27, and 56 because the level α V β 3 expression was known to be important in angiogenesis and the screening of various modulatory compounds for therapeutic purposes was conventionally used in the art at the time of the invention and the advantages of siRNA over other sequence specific modulators was well known in the art at the time the invention was made. Thus one of ordinary skill in the art would have had motivation and a reasonable expectation of success in making and using the claimed invention.

8. No claims allowed.
9. Applicants request for an interview is noted. However in view of the new grounds of rejection Examiner deemed it necessary to make the rejections part of the record before an

additional interview occurred so that Applicants could review the new grounds of rejection and fully respond to the them.

10. All other objections and rejections recited in Office Action of December 12, 2007 are withdrawn.

11. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Peter J. Reddig whose telephone number is (571) 272-9031. The examiner can normally be reached on M-F 8:30 a.m.-5:00 p.m.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Larry Helms can be reached on (571) 272-0832. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Peter J Reddig/
Examiner, Art Unit 1642

/P. J. R./

/Karen A Canella/
Primary Examiner, Art Unit 1643